Journal of
Cardiology & Cardiovascular
Surgery

Original Article

DOI: 10.51271/JCCVS-0043

Electrocardiographic indicators of atrial fibrillation in obstructive sleep apnea syndrome: a retrospective analysis of P-wave peak time

Oğuzhan Baran[1], Yücel Yılmaz[1], Mustafa Kaan Dişyapar[1], Nur Aleyna Yetkin[2], Fatma Özdemir[3], Şaban Keleşoğlu[4]

[1]Department of Cardiology, Kayseri City Hospital, Kayseri, Turkiye
[2]Department of Chest Diseases, Faculty of Medicine, Erciyes University, Kayseri, Turkiye
[3]Department of Chest Diseases, Kayseri City Hospital, Kayseri, Turkiye
[4]Department of Cardiology, Faculty of Medicine, Erciyes University, Kayseri, Turkiye

Cite this article: Baran O, Yılmaz Y, Dişyapar MK, Yetkin NA, Özdemir F, Keleşoğlu Ş. Electrocardiographic indicators of atrial fibrillation in obstructive sleep apnea syndrome: a retrospective analysis of P-wave peak time. *J Cardiol Cardiovasc Surg.* 2024;2(4):79-82.

Corresponding Author: Şaban Keleşoğlu, dr.s.k@hotmail.com

Received: 04/11/2024 ◆ **Accepted**: 13/12/2024 ◆ **Published**: 29/12/2024

ABSTRACT

Aims: Obstructive sleep apnea syndrome (OSAS) is a sleep-related breathing disorder characterized by hypopnea and apnea. Atrial fibrillation (AF) is an arrhythmia frequently encountered in cardiology practice, with concomitant heart disease and an increasing incidence with age. OSAS and AF are closely related as they have similar etiological risk factors and common pathophysiological processes such as inflammation, oxidative cellular damage, and autonomic nervous system dysregulation. P-wave peak time (PWPT) is the time from the beginning of the P-wave to its peak and is a recently defined electrocardiographic (ECG) parameter. Studies on the relationship between PWPT and cardiovascular events have been published recently. In this study, we aimed to evaluate the risk of AF in OSAS patients by determining a new ECG parameter, PWPT.

Methods: 52 OSAS patients and 41 healthy individuals as a control group were included in the study. The groups were compared in terms of demographic characteristics, laboratory findings, echocardiography and ECG findings. D2 and V1 leads were used for PWPT as recommended in the literature.

Results: When the patient group was compared with the control group, no difference was found in terms of demographic characteristics and laboratory findings. Compared with the control group, OSAS patients had significantly longer PWPT (PWPTV1 55.05msn±7.18 msn vs 48.91 msn±7.08 msn, p<.01, PWPTD2 54.13 msn±5.60 msn vs 46.20 msn±6.94 msn, p<.01).

Conclusion: We observed that PWPT was longer in OSAS patients than in controls, and our results suggest that OSAS patients are at risk of AF.

Keywords: Obstructive sleep apnea syndrome, atrial fibrillation, P-wave peak time

INTRODUCTION

Obstructive sleep apnea syndrome (OSAS) is a sleep-related breathing disorder characterized by hypopnea and apnea. These respiratory disorders cause increased respiratory effort, blood hypoxia, hypercapnia, repeated nocturnal awakenings, and intermittent hypoxia, which leads to increased sympathetic nervous activity.[1,2] The incidence of OSAS is 24% among men and 9% among women aged 30 to 60.[3,4] It is estimated that it affects approximately 1 billion people worldwide.[5] Polysomnography is the gold standard for diagnosis and uses the number of apneas and hypopneas per hour of sleep [apnea-hypopnea index (AHI)]. For OSAS diagnosis, AHI must be >5, and when AHI is ≥30, it is considered severe OSAS.[6]

The relationship between OSAS and cardiovascular disease (CVD) is well-documented, often associated with hypertension (HT), heart failure (HF), atrial fibrillation (AF), coronary artery disease (CAD), and stroke.[7,8] AF is a common arrhythmia, with its prevalence increasing alongside concomitant heart disease and advancing age.[9] AF is associated with increased mortality and morbidity (thromboembolic and cardiovascular events, decreased quality of life, decreased exercise capacity, and decompensated HF).[10] Although causality between OSAS and AF has not yet been established, epidemiological studies suggest that OSAS doubles the risk of AF.[6] Furthermore, AF recurrence after cardioversion is significantly higher in untreated OSAS patients than in well-treated OSAS patients.[11]

Electrocardiography (ECG) is the main diagnostic tool for the diagnosis and treatment of CVD, especially in ischemic and arrhythmic conditions. Studies have been conducted on the availability of standard ECG and P wave indices (due to AF mechanisms).

This work is licensed under a Creative Commons Attribution 4.0 International License.

P-wave peak time (PWPT) is the time from the beginning of the P wave to its peak. It is considered a new index of atrial depolarization, and recent studies have shown a relationship between PWPT and CAD severity, left ventricular end-diastolic pressure, absence of coronary reflux, and left atrial volume index (LAVI).[12-14] This new parameter may be used as an indicator of the risk of developing AF.[15,16] In this article, we aim to determine the PWPT times in patients with OSAS and evaluate the risk of AF in patients with OSAS based on this index.

METHODS

The study was conducted with the permission of Kayseri City Hospital Clinical Researches Ethics Committee (Date: 12.12.2023, Decision No: 957). All procedures were carried out in accordance with the ethical rules and the principles of the Declaration of Helsinki.

The study included patients diagnosed via polysomnography in the sleep laboratory of the chest diseases department between 2020 and 2022 (AHI >5 and previously untreated). The study was planned retrospectively and all medical histories, physical examinations, laboratory findings, 12-lead ECG and transthoracic echocardiography records were obtained from archive files and/or computer records. The control group comprised age- and gender-matched patients without respiratory disease suspicion, based on medical history, physical examination, and echocardiography findings.

In addition, none of the patients included in the study had a history of paroxysmal AF. Patients with uninterpretable ECGs (left bundle branch block, presence of pacemaker, U waves and negative T waves on pre-stimulus ECGs), history of ischemic heart disease, non-sinus rhythm and/or arrhythmia (atrial or ventricular) and pacemaker history, segmental or global wall motion abnormalities, moderate to severe valvular heart disease, structural heart disease, endocrine neoplasms, parathyroid cancer, thyroid cancer or hyperparathyroidism, renal failure, hypertrophic cardiomyopathy, severe valvular disease, hypokalemia and hyperkalemia, hypomagnesemia and hypermagnesemia, creatinine clearance (CrCl) <60 ml/min, and patients with severe comorbidities were excluded from the study.

Sleep Test

Polysomnographic evaluation was performed in the sleep laboratory by continuous monitoring and analysis of ECG, electroencephalogram, electromyogram, pulse oximetry, electrooculogram, nasal airflow, snoring, leg movements, thoracic and abdominal movements, and body position. Polysomnographic records were evaluated by computer-assisted manual scoring according to the criteria of the American Academy of sleep medicine by physicians experienced in sleep disorders and polysomnography. OSAS was defined as the number of apneic and hypopneic events per hour during sleep.[17] Apnea was defined as the absence of airflow for at least 10 seconds. Hypopnea was defined as the reduction of airflow with 4% oxygen desaturation lasting at least 10 seconds with subsequent arousal.

Electrocardiogram Analysis

All standard 12-lead ECGs were performed in the supine position and at rest using an ECG device (Philips brand) standardized to 1 mV/cm and 25 mm/s paper speed. All ECGs were scanned and transferred to personal computers. ECGs were magnified 5-fold and measured using an electronic caliper (Cardio Calipers software version 3.3; Iconico.com, Philadelphia, PA, USA) for the necessary measurements. To reduce inaccurate measurements, ECG assessments were conducted by two cardiologists who were blinded to clinical information.

The PWPT in lead D2 (PWPTD2) was measured as the time from the onset of the P-wave to its peak in lead D2, and the PWPT in lead V1 (PWPTV1) was defined as the time between the onset of the P-wave and the lower limit of negative deflection in patients with biphasic or pure negative P-wave morphology (**Figure**).

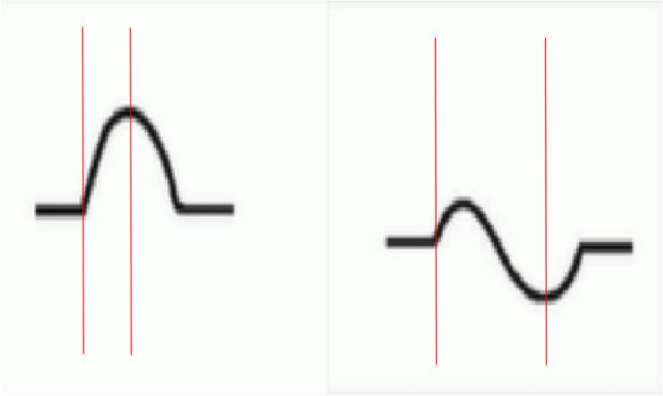

Figure. Measurement of P-wave peak time on electrocardiograms, **A.** Measurement of P-wave peak time in the lead D2 (positive wave), **B.** Measurement of P-wave peak time in the lead V1 (biphasic wave)
* Figure is used with permission from Yucel YILMAZ, MD.

Echocardiography

Patients and healthy volunteers underwent conventional echocardiographic examination with an M4S-RS (1.5-3.6 MHz) cardiac transducer and Vingmed System 5 (General Electronic Horten, Norway) echocardiograph. Left ventricular diastolic (LVIDd) and systolic (LVIDs) diameters, interventricular septum (IVSWT), and posterior wall (LVPWT) diastolic thicknesses were measured in the parasternal long axis with M-mode echocardiography according to the standards set by the American Echocardiography Association. The ejection fraction was calculated using the Teichholz formula.

Statistical Analysis

Statistical analyses were performed using the SPSS statistical software package for Windows version 21.0 (SPSS Inc., Chicago, IL, USA). The distributional characteristics of the data were determined using the Kolmogorov-Smirnov test. Independent samples t-test was used for parametric scale variables. Mann-Whitney U test was used for nonparametric scale variables. The $\chi 2$ (chi-square) test was used for the univariate analysis of categorical variables. Variables were expressed as mean±SD (standard deviation); categorical variables were expressed as percentages. A probability value of $p<0.05$ was considered to be significant, and two-tailed p values were used for all statistical analyses.

RESULTS

Baseline clinical and demographic characteristics of the study groups are presented in **Table 1**. There were no statistically

significant differences between the patient and control groups in terms of gender, age, smoking status, diabetes, and hypertension (p>0.05). In the patient group with OSAS, diastolic blood pressure was elevated and BMI was high (p<0.01).

Table 1. Baseline characteristics of the study populations

	OSAS (n=52)	Control (n=41)	p
Age (years)	52±9	51±4	NS
Gender (male/female)	35/17	27/16	NS
Systolic blood pressure (mmHg)	115±10.9	110±6	NS
Diastolic blood pressure (mmHg)	85±5.9	70±8	0,04
Stature (cm)	172±6	168±8	NS
Weight (kg)	89±17	70±7	<0,001
Body mass index (kg/m²)	30.24±5.9	24±2	<0,001

OSAS: Obstructive sleep apnea syndrome NS: Not statistically significant
Independent samples t-test was used for parametric scale variables. Variables were expressed as mean±SD (standard deviation); categorical variables were expressed as percentages.

The ECG parameters of the groups are shown in **Table 2**. Heart rate and QRS duration were similar between the groups (p<0.05). PWPTV1 and PWPTD2 were higher in OSAS patients compared to the control group (50.25±7 msec vs. 56.07±8.33 msec p<0.01 and 48.05±5.91 msec vs. 54.57±6.28 msec p<0.01, respectively).

Table 2. Echocardiography characteristics of the study population

Variables	OSAS (n=52)	Control (n=41)	p value
LVEDD (cm)	4.56±0.99	4.39±1	.367
LVESD (cm)	3.05±.86	3.41±1.01	.883
IVSD (cm)	0.9±.52	1.01±0.5	.625
PWD (cm)	0.92±0.29	1.01±0.6	.764
LVEF	62.1±3.7	63.9±3.8	.158

Data are expressed as mean±standard deviation for normally distributed data and percentage (%) for categorical variables. PHPT: Primary hyperparathyroidism, LVEDD: Left ventricular end diastole diameter, LVESD: Left ventricular end systole diameter, IVSD: Interventricular septal diameter, PWD: Posterior wall diameter, LVEF: Left ventricular ejection fraction

Table 3. Electrocardiographic characteristics of the study population

Variables	Control group (41)	OSAS (52)	p
Heart rate (beat/min)	79.3±7.6	81.7±9.7	.017
PR interval (ms)	141±14	145±16	0.891
PWPTV1 (ms)	48.91±7.08	55.05±7.18	**<.01**
PWPTD2 (ms)	46.20±6.94	54.13±5.60	**<.01**

PWPTD2: P-wave peak time obtained from D2 lead, PWPTV1: P-wave peak time obtained from V1 lead, Data are expressed as mean±standard deviation for normally distributed data and percentage (%) for categorical variables.

DISCUSSION

This is the first randomized study to show that PWPT prolongation detected by ECG analysis was higher in OSAS patients included in the patient group than in those included in the control group.

The pathophysiology of cardiovascular effects in OSAS can be explained as follows. Recurrent airway obstructions trigger hypoxemia, hypercapnia, intrathoracic pressure fluctuations, reoxygenation and sleep arousals.[8,18,19] All these recurrent attacks can activate various cardiovascular mechanisms such as vasoconstriction, tachycardia, acute blood pressure elevations and decrease in cardiac variability, which trigger sympathetic activation, and also increase in left ventricular wall stress, increase in afterload, acute diastolic dysfunction, left atrial stress, left atrial enlargement, hypercoagulability, oxidative stress and endothelial dysfunction can occur. Finally, all these mechanisms are associated with hypertension, systolic and diastolic dysfunction, coronary artery disease, sinus node dysfunction (sick sinus syndrome), atrioventricular block, AF, ventricular ectopy or even ventricular tachycardia and sudden cardiac death.[6]

AF is the most common arrhythmia in older ages and is associated with significant morbidity and mortality. Thus, primary prevention and identification of individuals at-risk have become more important. The atrial remodeling theory seems plausible to explain the predisposition to AF in patients with OSAS.[20-22] Sudden negative intrathoracic pressures may lead to repetitive atrial distension and gradual left atrial enlargement.[23] As a result of left atrial enlargement, there may be remodeling of the pulmonary vein ostia, a known site for initiating and propagating AF.[24]

Previous studies have shown that a P-wave terminal strength of >0.04 mm/s is an independent predictor of AF.[25] P-wave terminal strength is the algebraic product of the duration and depth of the negative terminal portion of the P-wave. Goda et al.[26] reported that PWTF has high sensitivity for estimating PAF in patients with acute ischaemic stroke. Although Baturova et al.[27] reported that PWTF may not be a good predictor of PAF in patients with acute ischaemic stroke, Öz et al.[16] and Çinar et al.[28] showed a significant relationship between PWPT and AF in patients with acute ischaemic stroke. Platonov et al.[29] showed that prolonged PWD is associated with a history of PAF. Yıldırım et al.[15] previously reported PWPT predicted paroxysmal AF in patients with a history of AF. In contrast, Burak et al.[14] found that only PWPT was associated with more extensive coronary atherosclerosis in patients with acute coronary syndrome. Yıldız et al.[12] showed that a prolonged PWPT was independently associated with an increased LAVI in hemodialysis patients. In our study, we found prolongation in PWPT in ECGs of patients with OSAS. When evaluated together with the literature, it can be assumed that prolongation of PWPT found in OSAS patients in this study will increase the risk of developing AF.

Limitations

The current study has some limitations. The study is a single-center, retrospective study with a limited number of patients. We do not know how long the patients had the disease before diagnosis. Although values such as PWPT are secondary markers of arrhythmia, we did not perform long-term clinical follow-up and rhythm Holter monitoring to detect the development of arrhythmia. Patients in our control group did not undergo sleep testing, which may have led to false results. OSAS patients were not grouped according to disease severity; further studies that include OSAS patients more proportionally to disease severity may yield different results. We also did not evaluate the relationship between AHI and atrial arrhythmia parameters.

CONCLUSION

In conclusion, the findings of this study suggest that PWPT, derived from ECG-a simple, easily measured, and cost-effective test-could serve as a predictive marker for AF risk in OSAS patients. More comprehensive and multicenter studies should be conducted to better analyze all possible predictors of AF and to make more robust recommendations for the future.

ETHICAL DECLARATIONS

Ethics Committee Approval

The study was conducted with the permission of Kayseri City Hospital Clinical Researches Ethics Committee (Date: 12.12.2023, Decision No: 957).

Informed Consent

All patients signed and free and informed consent form.

Referee Evaluation Process

Externally peer-reviewed.

Conflict of Interest Statement

The authors have no conflicts of interest to declare.

Financial Disclosure

The authors declared that this study has received no financial support.

Author Contributions

All of the authors declare that they have all participated in the design, execution, and analysis of the paper, and that they have approved the final version.

REFERENCES

1. Lévy P, Kohler M, McNicholas WT, et al. Obstructive sleep apnoea syndrome. *Nat Rev Dis Primers*. 2015;1(1):15015.
2. Lavie L. Oxidative stress in obstructive sleep apnea and intermittent hypoxia--revisited--the bad ugly and good: implications to the heart and brain. *Sleep Med Rev*. 2015;20:27-45.
3. Salzano G, Maglitto F, Bisogno A, et al. Obstructive sleep apnoea/hypopnoea syndrome: relationship with obesity and management in obese patients. *Acta Otorhinolaryngol Ital*. 2021;41(2):120-130.
4. Senaratna CV, Perret JL, Lodge CJ, et al. Prevalence of obstructive sleep apnea in the general population: a systematic review. *Sleep Med Rev.* 2017;34:70-81.
5. Benjafield AV, Ayas NT, Eastwood PR, et al. Estimation of the global prevalence and burden of obstructive sleep apnoea: a literature-based analysis. *Lancet Respir Med*. 2019;7(8):687-698.
6. Martí-Almor J, Jiménez-López J, Casteigt B, et al. Obstructive sleep apnea syndrome as a trigger of cardiac arrhythmias. *Curr Cardiol Rep*. 2021;23(3):20.
7. Marin JM, Agusti A, Villar I, et al. Association between treated and untreated obstructive sleep apnea and risk of hypertension. *JAMA*. 2012;307(20):2169-2176.
8. May AM, Van Wagoner DR, Mehra R. OSA and cardiac arrhythmogenesis: mechanistic insights. *Chest*. 2017;151(1):225-241.
9. Dilaveris PE, Gialafos EJ, Andrikopoulos GK, et al. Clinical and electrocardiographic predictors of recurrent atrial fibrillation. *Pacing Clin Electrophysiol*. 2000;23(3):352-358.
10. Chávez-González E, Donoiu I. Utility of P-wave dispersion in the prediction of atrial fibrillation. *Curr Health Sci J*. 2017;43(1):5-11.
11. Kanagala R, Murali NS, Friedman PA, et al. Obstructive sleep apnea and the recurrence of atrial fibrillation. *Circulation*. 2003;107(20):2589-2594.
12. Yıldız İ, Yildiz PÖ, Burak C, et al. P-wave peak time for predicting an increased left atrial volume index in hemodialysis patients. *Med Princ Pract*. 2020;29(3):262-269.
13. Çağdaş M, Karakoyun S, Yesin M, et al. The association between monocyte HDL-C ratio and SYNTAX score and SYNTAX score II in STEMI patients treated with primary PCI. *Acta Cardiol Sin*. 2018;34(1):23-30.
14. Burak C, Çağdaş M, Rencüzoğulları I, et al. Association of P wave peak time with left ventricular end-diastolic pressure in patients with hypertension. *J Clin Hypertens (Greenwich)*. 2019;21(5):608-615
15. Yıldırım E, Günay N, Bayam E, Keskin M, Ozturkeri B, Selcuk M. Relationship between paroxysmal atrial fibrillation and a novel electrocardiographic parameter P-wave peak time. *J Electrocardiol*. 2019;57:81-86.
16. Öz A, Cinar T, Kızılto Güler C, et al. Novel electrocardiography parameter for paroxysmal atrial fibrillation in acute ischaemic stroke patients: P-wave peak time. *Postgrad Med J*. 2020;96(1140):584-588.
17. Kapur VK, Auckley DH, Chowdhuri S, et al. Clinical practice guideline for diagnostic testing for adult obstructive sleep apnea: an American academy of sleep medicine clinical practice guideline. *J Clin Sleep Med*. 2017;13(3):479-504.
18. Latina JM, Estes NAM, Garlitski AC. The relationship between obstructive sleep apnea and atrial fibrillation: a complex interplay. *Pulm Med*. 2013;2013(1):621736.
19. Patel N, Donahue C, Shenoy A, Patel A, El-Sherif N. Obstructive sleep apnea and arrhythmia: a systemic review. *Int J Cardiol*. 2017;228:967-970.
20. Dimitri H, Ng M, Brooks AG, et al. Atrial remodeling in obstructive sleep apnea: implications for atrial fibrillation. *Heart Rhythm*. 2012;9(3):321-327.
21. Arzt M, Young T, Finn L, Skatrud JB, Bradley TD. Association of sleep-disordered breathing and the occurrence of stroke. *Am J Respir Crit Care Med*. 2005;172(11):1447-1451.
22. Lau DH, Mackenzie L, Kelly DJ, et al. Hypertension and atrial fibrillation: evidence of progressive atrial remodeling with electrostructural correlate in a conscious chronically instrumented ovine model. *Heart Rhythm*. 2010;7(9):1282-1290.
23. Otto ME, Belohlavek M, Romero-Corral A, et al. Comparison of cardiac structural and functional changes in obese otherwise healthy adults with versus without obstructive sleep apnea. *Am J Cardiol*. 2007;99(9):1298-1302.
24. Haïssaguerre M, Jaïs P, Shah DC, et al. Spontaneous initiation of atrial fibrillation by ectopic beats originating in the pulmonary veins. *N Engl J Med*. 1998;339(10):659-666.
25. Platonov PG. P-wave morphology: underlying mechanisms and clinical implications. *Ann Noninvasive Electrocardiol*. 2012;17(3):161-169.
26. Goda T, Sugiyama Y, Ohara N, et al. P-wave terminal force in lead v1 predicts paroxysmal atrial fibrillation in acute ischemic stroke. *J Stroke Cerebrovasc Dis*. 2017;26(9):19121915.
27. Baturova MA, Sheldon SH, Carlson J, et al. Electrocardiographic and echocardiographic predictors of paroxysmal atrial fibrillation detected after ischemic stroke. *BMC Cardiovasc Disord*. 2016;16(1):209.
28. Çinar T, Hayiroğlu Mİ, Selçuk M, et al. Evaluation of electrocardiographic P-wave parameters in predicting long-term atrial fibrillation in patients with acute ischemic stroke. *Arq Neuropsiquiatr*. 2022;80(9):877-884.
29. Platonov PG. Atrial conduction and atrial fibrillation: what can we learn from surface ECG? *Cardiol J*. 2008;15(5):402-407.